U. S. NAVAL SUBMARINE MEDICAL CENTER

Submarine Base, Groton, Conn.

REPORT NUMBER 660

A REVIEW OF
THE PATHOGENESIS OF SKIN BENDS

by

LT. W. Landon Dennison, Jr., MC, USN

Bureau of Medicine and Surgery, Navy Department
Research Work Unit MF099.01.01.08

Released by:

J. E. Stark, CAPT MC USN
COMMANDING OFFICER
Naval Submarine Medical Center

22 April 1971

UNCLASSIFIED
Security Classification

DOCUMENT CONTROL DATA - R & D

Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1 ORIGINATING ACTIVITY (Corporate author)
Naval Submarine Medical Center, Naval Submarine Medical Research Laboratory

2a. REPORT SECURITY CLASSIFICATION
Unclassified

2b. GROUP

3 REPORT TITLE
A REVIEW OF THE PATHOGENESIS OF SKIN BENDS

4 DESCRIPTIVE NOTES (Type of report and inclusive dates)
Interim Report

5 AUTHOR(S) (First name, middle initial, last name)
W. Landon DENNISON, Jr., LT, MC, USN

6. REPORT DATE	7a. TOTAL NO. OF PAGES	7b. NO. OF REFS
22 April 1971	15	31

8a. CONTRACT OR GRANT NO

b. PROJECT NO
MF099.01.01.08

c.

d.

9a. ORIGINATOR'S REPORT NUMBER(S)
NSMRL Report Number 660

9b. OTHER REPORT NO(S) (Any other numbers that may be assigned this report)

10 DISTRIBUTION STATEMENT
Approved for public release; distribution unlimited.

11 SUPPLEMENTARY NOTES

12. SPONSORING MILITARY ACTIVITY
Naval Submarine Medical Center
Box 600 Naval Submarine Base
Groton, Connecticut 06340

13 ABSTRACT

The literature concerning "skin bends" was searched, especially as regards the causes of this condition. This investigator concluded (by compilation of the theories from the literature and application of physiological and anatomical data) that gases reach the various layers of the skin by perfusion from the blood and by penetration from without. The route by which the gases reach the skin and the histological area in which they form bubbles is variable, but the site where they form is important in production of the various clinical varieties of "skin bends".

DD FORM 1 NOV 65 1473 (PAGE 1)
S/N 0102-014-6600

UNCLASSIFIED
Security Classification

14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Caisson disease						
Skin bends						
Physiology of diving						
Hyperbaric environment effects						
Physiology of the skin						

A REVIEW OF THE PATHOGENESIS OF SKIN BENDS

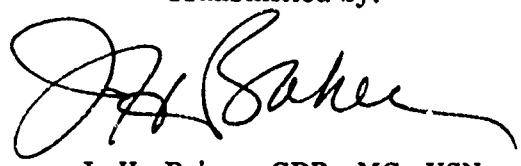
by

LT. W. Landon Dennison, Jr., MC, USN

NAVAL SUBMARINE MEDICAL RESEARCH LABORATORY
NAVAL SUBMARINE MEDICAL CENTER REPORT No. 660

Bureau of Medicine and Surgery, Navy Department
Research Work Unit MF099.01.01.08

Transmitted by:



J. H. Baker, CDR, MC, USN
Director, School of Submarine Medicine

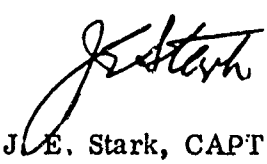
Reviewed and Approved by:

Charles F. Gell, M.D., D.Sc.(Med.)
Scientific Director
NavSubMedRschLab

Reviewed and Approved by:

J. D. Bloom, CDR, MC, USN
Officer-in-Charge
NavSubMedRschLab

Approved and Released by:



J. E. Stark, CAPT MC USN
COMMANDING OFFICER
Naval Submarine Medical Center

Approved for public release; distribution unlimited

SUMMARY PAGE

THE PROBLEM

To investigate the literature concerning the cutaneous aspects of decompression sickness, "skin bends", -- especially in regard to its causes.

FINDINGS

Most of the theories of the causes of "skin bends" have been derived from clinical observation, and little from experimental study. Conclusions by the present author were derived from evaluation of theories from the literature and application of physiological and anatomical data, namely (1) in the "dry dive", the skin is a sensitive indicator of the adequacy of decompression; and (2) differences in the appearance of the lesions may be due to their differences in etiology, viz endogenous and/or exogenous gases.

APPLICATIONS

Information presented in this report will be of use to submarine medical officers and persons concerned with hyperbaric medicine.

ADMINISTRATIVE INFORMATION

This report was prepared by a submarine medical officer candidate and submitted as partial fulfillment of the requirements for qualification as a submarine medical officer. It was selected for publication in order to make the information readily available in the School of Submarine Medicine and Technical Library at the Naval Submarine Medical Center.

The manuscript was approved for publication on 22 April 1971, and designated as NSMRL Report No. 660. It is report number 8 on the work unit MF099.01.01.

PUBLISHED BY THE NAVAL SUBMARINE MEDICAL CENTER

ABSTRACT

The literature concerning cutaneous aspects of caisson disease, "skin bends" was searched, especially as regards the causes of this condition. This investigator found (by evaluation of the theories from the literature and application of physiological and anatomical data) that gases reach the various layers of the skin by diffusion from the blood and by penetration by osmosis through the skin from the external ambient gas concentrate. The route by which the gases reach the skin and the histological area in which they form bubbles is variable, but the site where they form is important in production of the various clinical varieties of "skin bends".

A REVIEW OF THE PATHOGENISIS OF SKIN BENDS

INTRODUCTION

Many of those who have been exposed to extreme changes in atmospheric pressure, be it at high altitudes or the depths of the sea, may have experienced a mild case of pruritus. This dermatologic symptom will be called "skin bends" in this paper. Skin bends, a mild form of decompression sickness, has been variously called "les pouces" (fleas) by Paul Bert or "the itch" by the English speaking peoples. Most persons associated with diving limit the term "skin bends" to a transient "rash" with or without pruritus, however, this paper will consider all skin manifestations associated with decompression as "skin bends". The phenomenon has been known since men began to expose themselves to great alterations in barometric pressure[1].

Although it has been long recognized as an entity, little has been written specifically on skin bends, presumably because the signs and symptoms are fleeting and only mildly annoying. When the patient has other symptoms of decompression sickness, these symptoms are often severe, thus most injury reports list only a "rash" and then go on to describe the graver symptoms and signs. The intention of this paper is to attempt to determine the pathogenesis of the disease because it is felt that the skin, being exposed to both the diving medium and the blood, may react a little differently from the way other organs react to compression and decompression.

Skin manifestations of decompression sickness develop during or after decompression. Although it occurs both in aviators and deep-sea divers, the bulk of this paper will deal with deep sea divers or hyperbaric chamber subjects. Skin bends develop only in "dry dives" (with a few exceptions)[2]. A dry dive is one where the diver's skin is dry as in "hard-hat divers" with water tight suits or in hyperbaric chambers. SCUBA divers, on the other hand, have direct skin exposure to the water.

In Rivera's[3] analysis of decompression sickness taken from U. S. Navy files, 14.9 per cent of bends cases (935 total cases) had skin manifestations; Duffner's series 0.9 per cent; Behnke, 13.6 per cent; Thorne, working with caisson workers, found that 10 per cent of all bends cases developed had skin bends. Again, many more probably had very minor signs or symptoms which were not reported, and many of the divers in the above series had direct skin contact with water (as in SCUBA divers) and therefore would not develop skin bends anyway.

Skin bends take on several different forms. Aldao[4] lists six categories:

(1) Pruritus only - others[5] mention that a pricking sensation may also be present.

(2) Scarlatinaform rash - this is perhaps the most frequent sign. It is most frequent over the chest, shoulders, back, upper abdomen, and thighs, in

that order.[5,6] This form is usually accompanied by mild pruritus. The rash disappears within a few hours of surfacing and is not usually accompanied by more serious forms of decompression sickness.

(3) Erysipelas form - Has the same general distribution as the scarlatinaform rash. The lesions vary from small papules to very large plaques which cover the whole chest and shoulders. The borders are flat and firm and violet in color showing a characteristic venous network on the surface. Coughing or performing the Valsava maneuver will accentuate the venous markings (Mellinghoff's sign). This may accompany or precede the more serious forms of decompression sickness.

(4) Cutis marmorata marbleization--This is one of the danger signs in diving, because frequently this precedes major complications of decompression. Ferris and Engel in Fulton's Decompression Sickness[5] describe this lesion in great detail, from aviation studies, but there is no reason to suspect that the process differs from that in diving. They state that the lesion begins as a small pale area with cyanotic mottling. The lesion may or may not spread peripherally becoming erythematous with superimposed mottling. Continued exposure to relatively decreased atmospheric pressure causes spread over a large area. The temperature of the skin is 1-2° C. warmer in the erythematous stage. When recompressed the mottled area becomes diffusely red and hot and then disappears completely shortly after recompression is complete. However, 4-6 hours later the area becomes tender to palpation which increases in intensity for 24 to 36 hours, after which the tenderness decreases and is gone in two to three days. In most cases there is no change in the color of the skin during this period of tenderness, except for occasional erythema. Infiltration of the deep layers of skin with an anesthetic (subcutaneous tissues?) causes relief of the tenderness.

(5) Serious form--In this form the condition of the patient becomes totally cyanotic--here the patient is in shock and has signs of emboli in vital tissues. Aldao states a great quantity of blood is trapped in the cutaneous blood vessels, but he does not give any reference and furthermore states that he has never seen such a case.

(6) Emphysema:

(a) Intracutaneous--Ferris and Engel[5] describe minute intracutaneous blebs in aviators. These blebs are accompanied by mild pruritus and/or pricking sensations. This phenomenon has not been described in the diving literature seen by this author.

(b) Subcutaneous emphysema--Is seen frequently in air embolism associated with rapid ascent in the water and rupture of air into the pulmonary circulation or dissection of air into the neck and chest from pneumothorax. It is only seen in severe decompression sickness.

In further consideration of the pathogenesis of skin bends the fundamental of diving physiology and a review of skin anatomy and physiology

will follow, with special emphasis to the behavior of the skin in relation to gases. In research for this paper, the author has found little to explain the above phenomena; many explanations have been offered. In the ensuing sections these hypotheses will be reviewed and discussed.

FUNDAMENTALS OF DIVING PHYSICS AND PHYSIOLOGY

In order to present a clear discussion of this subject, it is important to have an understanding of the physics and physiology of compression and decompression[7]. Man on the earth's surface is continually exposed to the weight of the atmosphere above and around him. This atmosphere exerts a pressure of 14.7 pounds per square inch (psi) which is equivalent to the pressure exerted by approximately 33 feet of sea water.

When an individual is subjected to greater or lesser pressures, the atmospheric gases (or those of the diving medium) react according to the various physical gas laws. The gas laws, although important, do not act alone. The principles of dynamic equilibrium and solubility are important forces. Solubility of gases, especially the biologically inert gases such as nitrogen and helium, determine how much more of a gas will be dissolved into the body's tissues than would occur with the simple application of Henry's Law, and perhaps more important, where that gas will be dissolved. For instance, nitrogen is highly soluble in fatty tissues, thus a great quantity of nitrogen will be dissolved into the fatty portion of the organism (Table I), and since helium is less soluble than nitrogen it has certain advantages over nitrogen as the inert portion of the breathing mixture, among which are its lower narcotic effect and faster elimination from the body.

Therefore, in the process of diving, gases are driven and dissolved into the body as the atmospheric pressure is increased, the reverse occurs when the barometric pressure is reduced.

Decompression, the act of reducing atmospheric pressure must be performed carefully. If done too rapidly the gases, especially the inert ones, will come out of solution and form bubbles. Bubbles usually do not form until the partial pressure of the dissolved gases exceed the partial pressure of the ambient gases by a factor of two ("Haldane's 2:1 Ratio"). Once they are formed, they grow as the barometric pressure is reduced. The expanding bubbles cause symptoms, such as pain if they are in tendons and muscles and CNS symptoms result from bubbles arising in fatty tissue or vital blood vessels in those organs. Here one can see the importance of adequate blood supply (high capillary-tissue ratio and adequate venous drainage) to clear the tissue of gases approaching the point of super-saturation for a particular pressure.

Symptoms resulting from bubbles thus occurring result in similar syndromes known as Caisson's Disease, Decompression Sickness or simply as Bends.

Since the skin is exposed to these gas forces from within (the blood) and

Table I. Solubility Coefficients of Gases in Oil and in Water at 38°C. (from Behnke[29])

	Nitrogen	Argon	Helium	Oxygen
In olive oil	0.0667	0.1395	0.0148	0.0113
In water	0.0125	0.0262	0.0087	0.023
Ratio	5.24:1	5.32:1	1.7:1	5.0:1

from without (the atmosphere), the inert gases may originate from either source, i.e., by direct penetration in osmotic exchange through the integument or from vascular diffusion.[30, 31]

ANATOMY AND PHYSIOLOGY OF THE SKIN

Considering the skin in relation to the rest of the body, one should recall that it is the largest organ in the body by weight and its surface area measures about 1.7 square meters, which is one-twenty-fifth of the lung's surface area[8]. The function of this integument is that of protection; secondarily, it aids in temperature regulation and to a minor extent some excretory processes take place.

The skin[9] can be considered as a sheet covering the body, modified in certain places to accommodate particular functions and stresses. Grossly it is divided into the epidermis and dermis (Figure 1); the epidermis is the upper layer and is composed of five layers of cells (Figure 2). The outermost layer, the stratum corneum, is a compact, dead, horny layer undergoing constant desquamation. Covering this layer is a thin, oily layer of material secreted from the sebaceous glands. Below the stratum corneum one finds successively, the stratum lucidum, the stratum granulosum, the prickle cell layer, and lastly the stratum germinativum. The cells of the stratum germinativum (basal layer) are the metabolically active cells which migrate upwards as aging occurs to the layers above and are finally desquamated from the stratum corneum. The epidermal appendages (hair follicles, sweat glands and sebaceous glands) are seen as invaginations of epidermis into the dermis. These invaginations are very thin walled in comparison with the rest of the epidermis. The epidermis has no nerve or blood supply per se, but nutrition diffuses from the vessels of the dermis. Note that the dermal surface is not smooth but has many small elevations which protrude into the epidermis. The blood supply of the dermis is quite intricate (Figure 3), and since it is germaine to the subject of skin bends, it will be considered in detail.

(a) The cutaneous arterial network. This is a richly anastamosing network

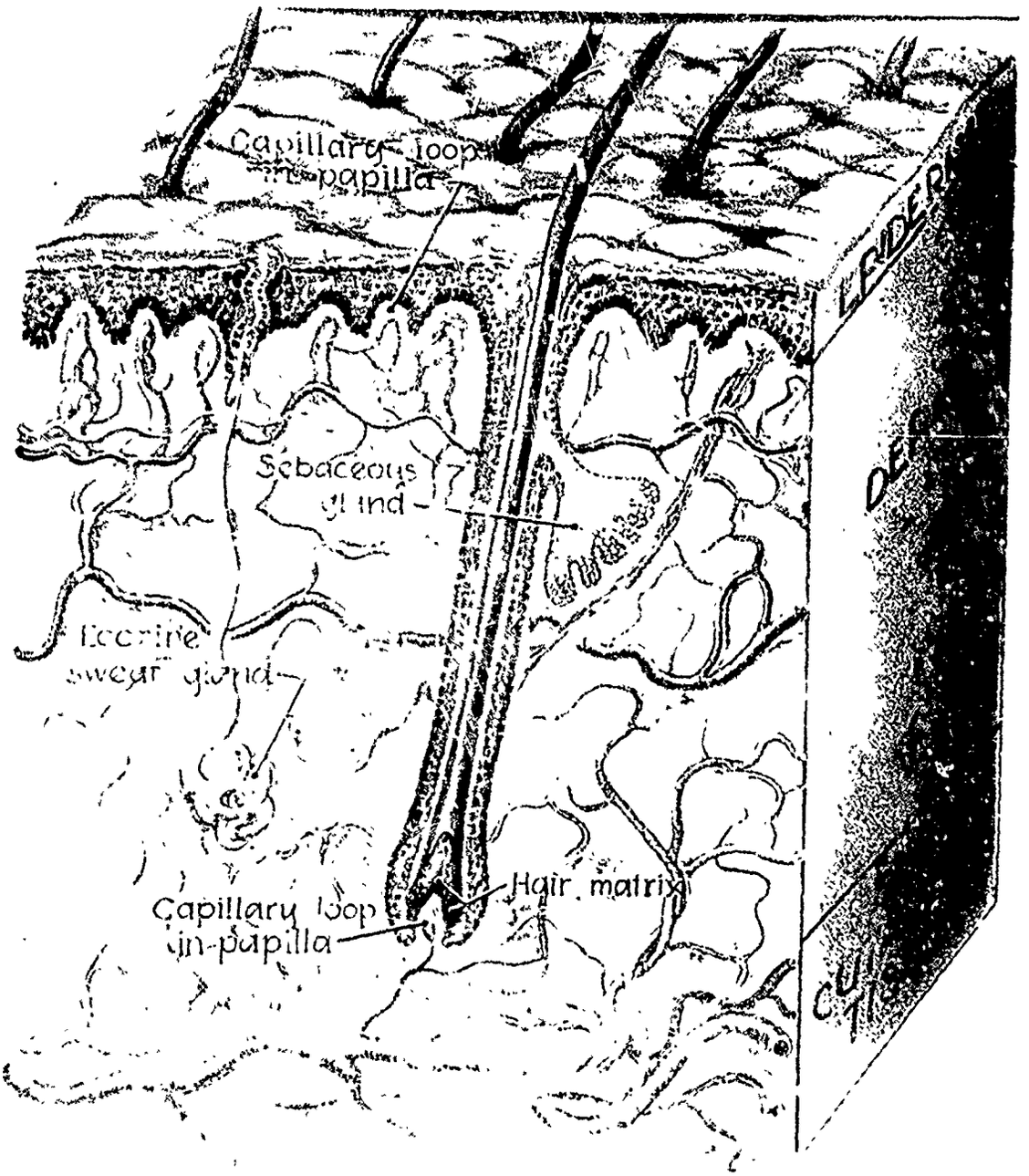


Fig. 1. The anatomy of the skin.

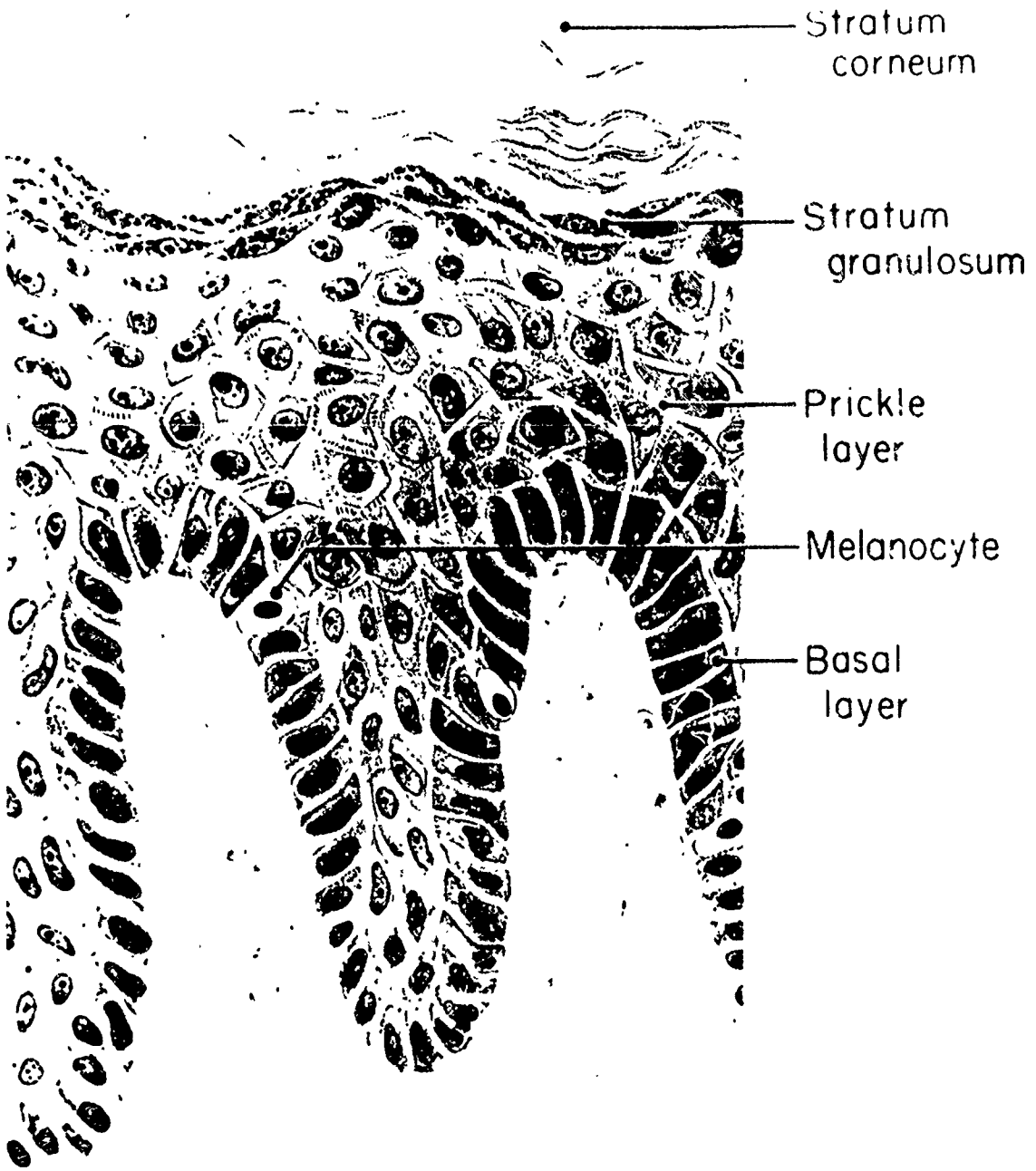


Fig. 2. Histology of the epidermis.

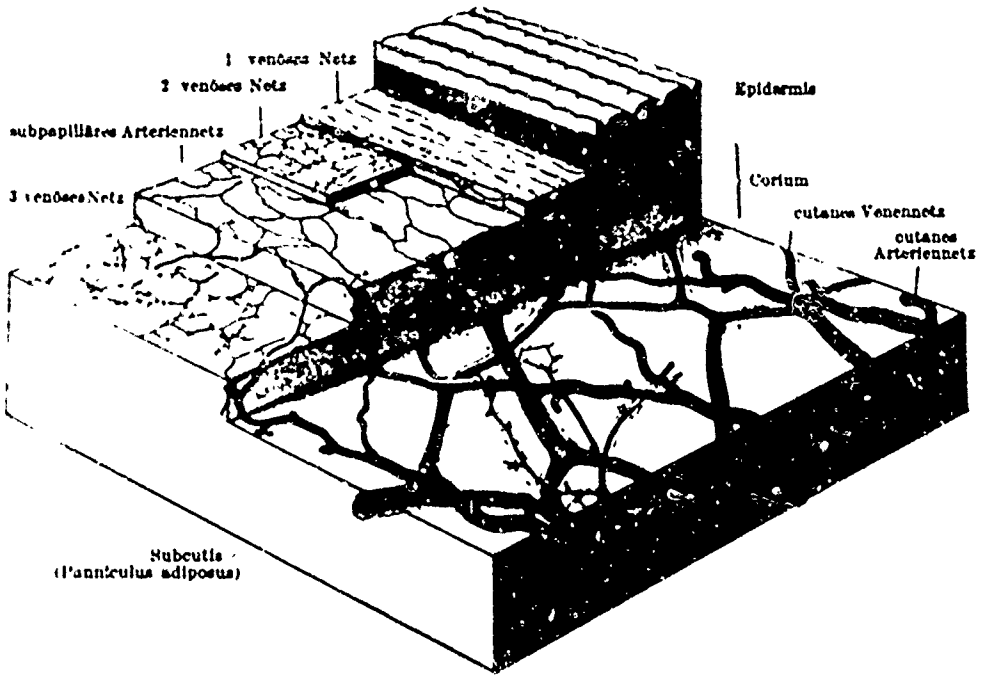


Fig. 3. A diagram of the skin and its vessels, showing the arrangement of the arterial and venous plexuses at various levels (after Spalteholz). From Spalteholz (261). (Reproduced by permission of Springer-Verlag.)

deep in the corium, from this, relatively straight arterioles ascend to the upper corium to form the sub papillary arterial plexus. This plexus has oblong meshes running parallel to the papillary ridges. Branching from the subpapillary plexus, smaller arterioles called the papillary arterioles supply the papillary capillary network. Much of the nutrition of the epidermis comes from these capillaries by diffusion.

(b) Venous circulation. The venous circulation roughly parallels the arterial circulation, but the venules are characteristically narrow until they descend into the depths of the corium where large meshes are formed. Between the dermis and the subdermis are large veins, many of which contain valves. Some of these veins are very thin-walled and a certain amount of exchange takes place through the walls of the veins.

(c) Anastomoses. The papillary arterioles anastomose with muscular venules. The muscular metaarterioles regulate the flow through the capillary bed.

Skin color depends largely on this circulation. The predominant role is played by the subpapillary venous plexus. Table II shows the skin color in various circulatory states.

The nerve supply of the skin is rich, yet no nerves enter the epidermal layer; all nerve endings are in the upper levels of the corium and around the epidermal appendages. The nerves are very responsive to noxious stimuli, both local and general. Alterations of the environment are often reflected in the local circulation, which in turn changes the skin color or appearance, a good example of which is the Triple Response of Lewis.

Chemically, the integument of the human body complements the role of protection. The granular layer contains granules of keratohyaline and eleidin which are probably the beginning of transformation of the protoplasm of the cell to the waxy and fatty material with which the surface cells are loaded. Sebaceous glands supply a thin lipoidal film which covers the stratum corneum and hair.

Table II. Comparison of Skin Color with Cutaneous Circulation (from Krogh[11])

Skin Color	Arterioles	Capillaries	Venules	Blood Flow
Red	dilated	dilated	dilated	normal
Scarlet	normal	normal	normal	increased
Lt. pink	constricted	constricted	constricted	increased
White	constricted	constricted	constricted	decreased
Deep Blue	constricted	dilated	dilated	decreased

This film is composed of free and esterified fatty acids and unsaponafiable material.

Thus we see that the skin is a well designed armour protecting the "milieu interieur", however there appears to be some chinks in this armour, which will be considered in more detail.

PERMEABILITY OF THE SKIN

Generally, it can be stated that the skin is relatively impermeable to most substances, except for fluids and some vapors[9, 12]. The way in which gases penetrate the skin has been studied in some detail with particulate substances and liquids; but little is known in regard to how gases penetrate the skin. MacKee,[9] using heavy metals as tracers, found that the route of penetration was through the follicular pores and follicles and thence into the corium via the walls of the sebaceous glands and follicles. The metals then diffused from the corium in all directions. The exact layer of the skin which acts as the barrier to penetration is now known. The stratum corneum does not act as a barrier. The closely packed layers of the stratum granulosum is the area where most of the barrier action takes place. This is known as the Szakall layer.

Many authorities[9, 12, 26] state that fluids do not necessarily follow the route as outlined by MacKee, but merely diffuse through the epidermis as gases diffuse through any other membrane. Scheuplein[13] recently reported that his data from electron microscopy and diffusion studies indicated that liquids diffused through the cells rather than around them. Gas diffusion is dependent on concentration on both sides of the membrane, molecular size, temperature and solubility.

Behnke[14], found that complete nitrogen desaturation could not occur during oxygen breathing because the entrance of nitrogen through the skin occurred at a rate of about 15-20 cc. per hour. Diffusion of nitrogen would increase if the temperature were increased or if an incision were made in the skin. In another report, Behnke and Willmon[15] discovered that helium was absorbed at a rate of 170 cc. per hour. These findings have been substantiated in more recent experiments by Klocke[16] who also correlated the increased diffusion with an increase in cutaneous blood flow. Shaw and Messer[17] and Chambers and Goldschmidt[18] have made some interesting observations which postulate a dual blood supply for the skin--persons enclosed in a box filled with nitrogen developed an increase in the respiratory uptake of oxygen.

When one considers the changes that occur about the skin when the atmospheric pressure is increased, we can say that grossly, the uptake of gas through the skin is enhanced on compression and that the release of gas from the skin during decompression is variable, viz. cooling decreases gas transfer, because of decreased cutaneous circulation, but increased because of the decreased exterior concentration of the gas. Whether the skin is wet or dry probably makes some difference; radon was found to pass through the skin faster when the subject

was in a water bath, in the same experiments radon penetrated the skin faster when grease was applied to the skin[9].

Thus, it is seen that there are many variables and unknowns in this problem, but with this information, one has better insight into the problem of skin bends.

THEORIES ON THE CAUSE OF SKIN BENDS

The author has found very few histological descriptions of skin bends, and those that have been found are usually of massive decompression sickness, where gross subcutaneous emphysema and severe gas embolization of the larger blood vessels were found. Nothing has been found regarding the less serious, less spectacular lesions.

Most of the proposed explanations for production of skin bends fall into three categories: (1) Expansion of gases in the sebaceous glands and sweat glands. (2) Gaseous embolization of blood vessels and (3) Reflex vasoconstriction, vasodilatation and piloerection due to trauma in the skin and subcutaneous tissues.

Paul Bert, in his classic Barometric Pressure[1], was of the opinion that the "fleas" (itch) was due to irritation of the tissues by fine bubbles even if the circulation was not stopped. Hill and McLoed[19] wrote that they had difficulty applying the cupping glass when treating patients with Caisson's Disease, concluding that great amounts of dissolved gases were present in the skin. Hill and Greenwood[20] in another paper felt that the rash after decompression was due to emoblization of vessels in the subcutaneous fat. But Erdman[21] when commenting on Hill's findings, noted that the rash was more frequent when sweating was less free, and therefore concluded that the eruption was due to the expansion of gases in the sebaceous glands. This opinion was also voiced by Shilling.[22] Behnke[14] and Anthony[23] also noted that rash and pruritus occurred with regularity if the skin is chilled during decompression. Harvey[27] in experimental studies, found that vasoconstriction favored bubble formation, although Duffner[4] concluded that bubbles in the capillaries stimulated the contractile cells of the capillaries.

Rashbass[2] conducted a series of experiments to determine the etiology of the itch, which was the only experimental publication found on this subject. He conducted a series of chamber dives to a simulated depth of 240 feet for 18 minutes. This dive produced the itch in all subjects, but no itch occurred when a portion or all of the body was immersed in water. This indicated that gas entered the skin directly from the outside. Repeated exposure using barrier creams, glycerine, etc. showed no effect on the severity of the pruritus. The portions of the body with the highest sebaceous content were the most common sites for the rash or itch. It should be noted that the decompression schedule which Rashbass used differs from that of the U.S. Navy Schedules[7]: (1) The depth and time falls into the category of Exceptional Exposure and (2) The stage decompression was considerably shorter than called for in the U.S. Navy

Decompression Tables for Exceptional Exposures. This confirms the opinion of others[26] that the rash and itch is present more frequently when marginal decompression is used. Behnke[24] has noted that bubbles have been seen in the cutaneous arteries and veins after rapid decompression. Aldao[4] states that all the eruptions seen are a manifestation of bubbles in the cutaneous vessels, and Wright et al[25] surmise the mottling of the skin is due to capillary embolization with gas.

At this juncture we find fairly general agreement on the fact that the "rashes" (which probably represent some of those descriptive entities mentioned, viz: pruritus scarlatinaform rash, and intracutaneous emphysema) are manifestations of expansion of gas which has entered the body from the outside directly into the skin, the sebaceous glands or sweat glands. Ferris and Engel have written some interesting thoughts that may account for formation of the other types of skin bends. Writing about cutis marmorata, they state that the pale, cool, cyanotic mottling that occurs is caused by the obstruction of terminal arterioles and venules. The transformation to erythema suggests that the obstruction is overcome by vasodilatation, akin to reactive hyperemia. The concentric spreading of the cutis marmorata may be secondary to a spreading factor in tissues, since the spread cannot be explained anatomically. The deep pain which is present following the disappearance of the marbleization suggests trauma as the potential spreading factor. The trauma is probably due to bubble formation in the subcutaneous fat.

Thus the theories for production of skin bends are varied, and probably all of them are functioning to some extent at one time or another. In summary then, we have the following propositions on rash formation:

(1) Expansion of gases in the sebaceous and sweat glands.

(2) Blood vessel gas thrombosis.

(3) Reflex changes in the blood vessels, secondary to trauma of gas expansion.

The following discussion will deal with the facts heretofore presented.

DISCUSSION

To review some of the facets covered thus far, in the pathogenesis of skin bends one must consider the various alterations in the diver's environment: Increased air pressure, dissolution of gas into the tissues, especially highly soluble gases such as nitrogen, chilling during decompression with subsequent vasoconstriction and release of gases from the tissues. Bends do not usually occur until Haldane's 2:1 ratio is exceeded, although this may not be true with skin bends, i.e., the ratio may be lower. What then, is the cause of skin bends? Rashbass's work leads one to suspect that the itching and the transient rashes are due to the rapid expansion and evolution of gases which originated from the outside. However, the observations of Ferris and Engel on the spread of cutis marmorata point to trauma deep in the skin which would

make one suspect that the trauma was from bubbles expanding in the loose connective tissue of the corium or subcutaneous tissues. Probably both etiologies are correct, and that exogenous and endogenous gases have a role in the production of different morphologic changes.

The author proposes the following as the etiology of the various manifestations of skin bends, these proposals are not proven but seem logical in view of the findings already presented.

(1) Skin bends are produced by the expansion of both exogenous and endogenous gas bubbles.

(2) The morphological pattern of the lesion depends largely on where the greatest quantity of gas is present (the initial lesion).

(3) Subsequent to the initial lesion (#2.) secondary vascular reaction may predominate.

Using the above proposals, let us examine the different categories of skin bends as outlined by Aldao.

(1) Pruritus alone: this probably represents small amounts of gases in the very superficial layers of the dermis, the irritant* is probably not present in large enough quantities or present long enough to cause secondary vascular reaction, but is deposited in an area supply by nerves, i.e., below the epidermis. The frequency of the symptom over the areas of high sebaceous gland content leads one to conclude that the dermis is reached via the sebaceous glands.

(2) Scarlatinaform rash: In all likelihood, this is an extension of the process which occurs in pruritus alone. The irritant gas is present in greater concentration and takes longer for it to evolve so that secondary vascular reaction occurs as well as stimulation of the pilo-erectory apparatus and perhaps some tissue damage occurs with release of histamine.

(3) The erysipelas form leads into the category of endogenous gases as the etiologic source. Here the vascular pattern and the positive Mellinghof's sign indicate that the deeper veins are involved either directly by gaseous embolism or because of reflex constriction of the musculature of the veins, due to irritation elsewhere. It is not likely that enough gas from an exogenous source could wholly account for this type of lesion.

(4) Cutis marmorata: Since this is seen only with severe bends, the source of the gas is endogenous and here bubbles are formed either in the tissues or in the adjacent blood vessels. This is a cutaneous manifestation of what is or will be occurring in the rest of the body.

(5) The serious form: Again, this is most likely an extension of the marbleization phenomenon.

(6) Emphysema: The intracutaneous forms described in aviators represents expansion of gas below one of the major "skin barriers" such as the Szakall

*"Irritant" does not imply that the gas is chemically offensive, rather, a gas bubble is the "irritant."

layer. Subcutaneous emphysema is due to either pneumothorax or massive decompression sickness where gas is found in all the organs and tissues of the body.

In conclusion, then, the skin appears to be a sensitive indicator of the adequacy of decompression, and when inadequate this is reflected by changes in the skin. These changes appear to be due to gas bubbles whose origin is gas induced osmosis from the surrounding medium and diffusion from the blood.[30,31]

Sci. Dir.'s Note: In March 1970, at the F. C. Hall Environmental Laboratory, Duke University Medical Center, Durham, North Carolina, during a "saturation" dive at a simulated depth of 200 feet in a gaseous environment of helium and oxygen,[30] all three experimental subjects developed urticaria during or shortly after breathing a normoxic nitrogen gas mixture. To experienced observers these cutaneous symptoms and signs were similar to those frequently observed following decompression, and therefore considered a manifestation of decompression sickness. However, no decompression preceded the above symptomatic reactions, suggesting an association of the urticaria with some inherent difference in the inert gases such as a physical property whose effect upon the tissue is enhanced under hyperbaric conditions. As stated before, the lesions occurred before the subjects had experienced decompression, and although they appeared very similar to the cutaneous reactions frequently termed "skin bends", they were not observed upon skin within the head-tent and hence over those areas exposed to the breathing mixture at all times. The urticaria has therefore been attributed to some cause arising from the concentration gradient maintained between cutaneous blood and chamber atmosphere in the affected areas rather than considered a manifestation of decompression sickness. Gas-induced osmosis is discussed as a possible initiating mechanism and is shown to be quantitatively consistent with the clinical observations. This evidence appears to support the author of this report in his contention that there is an exterior gaseous osmotic exchange in the skin with subsequent gas expansion as a contributing factor to the urticaria. Similar lesions were noted under the same conditions on subjects in the recent saturation dives conducted at the University of Pennsylvania Medical School Environmental Institute, although the theoretical observation as to the cause of the lesions has not been defined to the satisfaction of the University of Pennsylvania investigators.

CONCLUSIONS

Little experimental evidence was found to account for the phenomenon of skin bends, and most of the conclusions were drawn from the clinical appearance of the lesions. This author concludes that certain types of skin bends are caused by expansion of gases that were dissolved into the skin during compression and that other lesions were due to expansion of gases that were delivered to the tissues via the blood stream. The patterns of the eruptions vary depending on secondary vascular reaction.

Further investigation on the physiology of the skin during hyperbaric conditions is indicated.

REFERENCES

1. Bert, P.: Barometric Pressure. Researches in Experimental Physiology (1878) Hitchcock, M. A. and Hitchcock, F. A., trans. College Book Co., Columbus, 1943.

2. Rashbass, C.: The Aetiology of Itching on Decompression, Royal Navy Physiologic Laboratory Reports, 1957.

3. Rivera, J.C.: Decompression Sickness Among Divers: An Analysis of 935 Cases, EDU Research Report 1-63, February 1963.

4. Aldao, C.N.G.: Cutaneous Forms of Caisson's Disease, Rev. Argent. Dermatosif., 33: 20-25, 1949.

5. Ferris, E. B. and Engel, G. L.: p35 in Decompression Sickness, John F. Fulton, Ed., W. B. Saunders Co., Philadelphia, 1951.

6. Golding, F. C.: Decompression Sickness Occurring During the Construction of the Dartford Tunnel, Brit. Jour. Indust. Med., 17: 167, 1960.

7. U. S. Navy Diving Manual (NAVSHIPS 250-538), Bureau of Ships, Navy Department, G. P. O., July, 1963.

8. Best, C. H. and Taylor, N. B.: The Physiologic Basis of Medical Practice, Williams and Wilkins Co., Baltimore, 1955, p345.

9. Rothman, S.: Physiology and Biochemistry of the Skin, Univ. of Chicago Press, 1954, Chicago.

10. Duffner, G. J.: The Capillary Circulation - Its Role in Compressed Air Illness. Submarine Medical Officer Qualification Thesis.

11. Krogh, A.: The Anatomy and Physiology of the Capillaries, Yale Univ. Press, 1924 (quoted from Duffner (10)).

12. Pillsbury, D. M., et al: Dermatology, W. B. Saunders Co., Philadelphia, 1956.

13. Scheuplein, R. J. Routes of Penetration Through the Skin from Permeability Data. Report read to the Society of Investigative Dermatology, Inc., 20 June 1965, New York City.

14. Behnke, A. R.: The Absorbtion and Elimination of Gases of the Body in Relation to its Fat and Water Content, Medicine, 24: December, 1945.

15. Behnke, A. R. and Willmon, T.L.: Cutaneous Diffusion of Helium in Relation to Peripheral Blood Flow and Absorbtion Atmospheric Nitrogen Through the Skin, Am. J. Physiol., 13: 627-632, 1941.

16. Klocke, R. A., et al: Gas Transfer Across the Skin in Man, J. Appl. Physiol., 18: 311-317, 1963.

17. Shaw, L. A. and Messer, A. C.: Cutaneous Respiration in Man: II. The Effects of Temperature and Humidity upon the Rate of CO_2 Elimination and Oxygen Absorbtion, Am. J. Physiol., 90: 107-118, 1929.

18. Chambers, A.H. and Goldschmidt, S.: The Influence of Atmospheric Oxygen Absorbtion upon Transparent Total Oxyger Utilization of the Body, Am. J. Physiol., 125: 331-332, 1940.

19. Hill, L. and McLoed, J., Jr.: Caisson's Disease and Diver's Palsy; Experimental Study, J. Hygiene, 3: 401-445, 1903.

20. Hill, L. and Greenwood, M.: Influence of Increased Barometric Pressure on Man, Proc. Roy. Soc., 77: 442-453, 1906.

21. Erdman, S.: The Acute Effects of Caisson's Disease, Int. Cong. Hyg. and Demog., 3: 619-625, 1913.

22. Shilling, C. W.: Compressed Air Illness, U. S. Naval Med. Bull., 36: 235-259, 1938.

23. Anthony, R., et al: Temperature and Decompression Sickness, National Research Council Report No. 136, 1943.

24. Behnke, A. R.: A Review of Physiological and Clinical Data Pertaining to Decompression Sickness, NMRI Report No. 4, 1947.

25. Wright, W. and Brady, J. W. S.: Compressed Air Illness or Caisson's Disease, in Billings-Forchheimer's Therapusis of Internal Diseases, George Blumer, ed., Appleton and Co., New York 1929.

26. Van der Aue, O. E., Keller, R. J. and Brinton, E. S.: E.D.U. Report No. 1, March 1949.

27. Harvey, E. N., et al: The Mechanism of Bubble Formation in the Blood of Animals in Relation to Decompression Sickness, National Research Council Report No. 229, 1943.

28. Calvery, H. O., et al: The Metabolism and Permeability of Normal Skin, Physiological Reviews, 26: 495-540, 1946. (This is an excellent review of the physiology of the skin).

29. Behnke, A. R. and Stephenson, C. S.: Applied Physiology, Annual Review of Physiology, Vol. IV, 1942.

30. Blenkarn, G. D., Aquadro, C., Hills, B. A. and Saltzman, H. A.: Urticaria Following the Sequential Breathing of Various Inert Gases at a Constant Ambient Pressure of 7 ATA: A Possible Manifestation of Gas Induced Osmosis, Aerospace Med., 42: 141-146, Feb. 1971.

31. Unpublished data.